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HEALTH EFFECTS ASSESSMENT FOR SULFURIC ACID

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Office of Research and Development
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16. ABSTRACT

This document represents a brief, quantitatively oriented scientific summary of health effects data. It was developed by the Environmental Criteria and Assessment Office to assist the Office of Emergency and Remedial Response in establishing chemical-specific health-related goals of remedial actions. If applicable, chemical-specific subchronic and chronic toxicity interim acceptable intakes are determined for systemic toxicants, or q_1^* values are determined for carcinogens for both oral and inhalation routes.

Since the documented effects of sulfuric acid relate solely to the portal of entry following inhalation exposure, interim acceptable intakes are not estimated. The document does present data concerning estimates of acceptable air exposure concentrations.

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PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with sulfuric acid. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the Chemical Abstracts, TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to September, 1984. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) source has been extensively utilized:

U.S. EPA. 1982. Air Quality Criteria for Particulate Matter and Sulfur Oxides. Vol. II. Environmental Criteria and Assessment Office, OHEA, Research Triangle Park, NC. EPA 600/8-82-029b. NTIS PB 84-156793.

The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the variable data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, the AIS or acceptable intake subchronic, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan). This type of exposure estimate has not been extensively used or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for AIS estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available. Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure.

The AIC, acceptable intake chronic, is similar in concept to the ADI (acceptable daily intake). It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980) for a discussion of this concept]. The AIC is route specific and estimates acceptable exposure for a given route with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for ranking reportable quantities; the methodology for their development is explained in U.S. EPA (1983).

For compounds for which there is sufficient evidence of carcinogenicity, AIS and AIC values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980). Since cancer is a process that is not characterized by a threshold, any exposure contributes an increment of risk. Consequently, derivation of AIS and AIC values would be inappropriate. For carcinogens, q_1^*s have been computed based on oral and inhalation data if available.

ABSTRACT

In order to place the risk assessment evaluation in proper context, refer to the preface of this document. The preface outlines limitations applicable to all documents of this series as well as the appropriate interpretation and use of the quantitative estimates presented.

Although considerable data are available concerning the effects of sulfuric acid, reported effects include only localized irritant effects at the point of entry following inhalation (respiratory tree and lungs). Data regarding systemic toxic effects are lacking. Therefore, an estimate of AIS or AIC, which by definition estimate absorbed dose in mg/day in relation to systemic toxicity, would be inappropriate. Available data, however, do allow estimation of an acceptable air exposure concentration of 0.07 mg/m^3. A CS of 26.8 was calculated for the effects of dental etching and erosions that occur rapidly at relatively low atmospheric concentrations.

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LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
AIC	Acceptable intake chronic
AIS	Acceptable intake subchronic
CAS	Chemical Abstract Service
CS	Composite score
NOEL	No-observed-effect level
TLV	Threshold limit value
TWA	Time-weighted average

1. ENVIRONMENTAL CHEMISTRY AND FATE

Sulfuric acid (CAS No. 7664-93-9), H_2SO_4, is a colorless, viscous liquid with a molecular weight of 98.08 and a specific gravity of 1.8357 (Donovan and Salamone, 1983). Sulfuric acid is soluble in water in all proportions with evolution of heat and it has a vapor pressure of 1 mm Hg at 145.8°C (Weast, 1980).

The primary sources of sulfuric acid in the atmosphere are vehicular emissions, combustion sources, explosive manufacture, furnace soot, sulfuric acid manufacture, steel manufacture and volcanic emissions (Graedel, 1978). Sulfuric acid is present in the atmosphere in the form of aerosols. The size fractionation of sulfur-containing aerosols [which primarily consists of H_2SO_4 and $(NH_4)_2\ SO_4$] show that in dry weather the aerosol is found in sub-0.65 μm particle size fractions, while under humid conditions, the aerosol is found in the 0.65-3.6 μm particle size range (U.S. EPA, 1982). Size fraction analysis of sulfate aerosol in the atmosphere also showed that the predominant amount (70%) of sulfate ions are present in the submicron particle range (U.S. EPA, 1982).

The fate of sulfuric acid aerosols in the environment has been studied by a limited number of investigators (U.S. EPA, 1982) and significant data gaps exist in this area. It is likely that sulfuric acid aerosol will react chemically with other species in the atmosphere. The source of inorganic sulfates in the atmosphere, particularly ammonium sulfate, has been demonstrated to be due primarily to chemical reactions in the atmosphere (U.S. EPA, 1982). Similarly, depending upon the amount of dilution by the moisture in the atmosphere, sulfuric acid aerosols may react with organics in the atmosphere to form sulfonates.

Besides chemical transformation, sulfuric acid aerosols in the atmosphere are likely to be removed through wet and dry deposition; however, no estimate of the half-life of atmospheric sulfuric acid due to its chemical transformation and physical removal processes was located in the available literature.

In aquatic media of pH >7, sulfuric acid reacts with carbonate, bicarbonate or hydroxides in the sediment or suspended particles, with the formation of sulfates. Since the majority of sulfates, with the exception of lead and calcium, are soluble in water, this reaction may remobilize the precipitated metals from the aquatic phase and decrease the pH of the solution. In aquatic media of pH <7, at least a part of the sulfuric acid may remain ionized in solution and may be mobile.

The majority of sulfuric acid in soils is expected to be removed by reaction with inorganic minerals or organic matter in soils. The extent of this chemical reaction may depend on the strength of the sulfuric acid in the soil. As the concentration of the acid decreases through reactions or dilution through water, the reactivity should also decrease. In highly sandy soil, sulfuric acid probably leaches into groundwater.

2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS

2.1. ORAL

Pertinent data regarding the absorption of sulfuric acid from the gastrointestinal tract could not be located in the available literature. Since the major toxic effect of sulfuric acid is local irritation, and because of the buffering capacity of the blood, it is unlikely that significant systemic exposures occur.

2.2. INHALATION

Pertinent data regarding the absorption of inhaled sulfuric acid could not be located in the available literature. Since the major toxic effect of sulfuric acid is local irritation and since inhaled sulfuric acid is largely neutralized by NH_3 in the expired air, it is unlikely that significant systemic exposure to sulfuric acid occurs.

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

Carson et al. (1981) reviewed more than 400 studies on the effects of sulfuric acid on humans and experimental animals. A thorough analysis of these data is not possible within the constraints of the current project. Therefore, the following review will concentrate on those studies that provide data on the effects of near threshold doses of sulfuric acid (Table 3-1).

3.1. SUBCHRONIC

3.1.1. Oral. Pertinent data regarding the subchronic oral toxicity of sulfuric acid could not be located in the available literature.

3.1.2. Inhalation. Schlesinger et al. (1978, 1979) reported that exposure of donkeys to ~0.1 mg H_2SO_4/m^3 (0.102-0.106 mg/m^3) for 1 hour/day, 5 days/week, for 6 months resulted in erratic bronchial clearance in the last week of exposure. Respiratory rates were reduced in all four animals, and two of the four donkeys sustained impairment of clearance with erratic improvement during a 3-month follow-up period. Concentrations as low as 0.071 mg/m^3 administered as "repeated doses" were reported to decrease the rate of bronchial mucociliary clearance, but the duration of these exposures was not reported.

Loscutoff et al. (1978) exposed dogs to atmospheres containing 1.0 mg H_2SO_4/m^3 for "repeated doses." The number of animals, sex, duration and number of exposures were not reported. No effects were reported and all exhaled sulfate was in the form of an ammonium salt.

Rats appear to be relatively insensitive to the effects of sulfuric acid aerosols. Lawkowski et al. (1979) exposed groups of 10 male Sprague-Dawley rats to ambient atmospheres or atmospheres containing 2.37 mg H_2SO_4/m^3,

TABLE 3-1

Threshold Doses for Sulfuric Acid Toxicity*

Species	mg H_2SO_4/m^3 (particle size)	Exposure Conditions	Length of Exposure	Length of Study	Comments	Reference
Monkey	~0.1 plus fly ash and/or SO_2 (NR)	22-24 hours/day	18 months	18 months	NOEL for some combinations of pollutants	Alarie et al., 1975
Donkey	0.102 or 0.106 (0.5 μm)	1 hour/day; 5 days/week	6 months	6 months	Sustained impairment in bronchial mucociliary clearance in two of four donkeys. Some improvement within 3-month recovery period.	Schlesinger et al., 1979
Donkey	0.071-1.364 (0.3-0.6 μm)	1 hour by nasal catheter	NR	NR	Bronchial mucociliary clearance slowed.	Schlesinger et al., 1978
Dog	1.0 (NR)	"repeated doses"	NR	NR	NOEL (at 3.5 mg/m³), increased pulmonary resistance.	Lesculoff et al., 1978
Dog	~0.09 in auto exhausts (NR)	16 hours/day	68 months	104 months	Emphysematous and other microscopic lung changes 3 years after 68-month exposure.	Lewis et al., 1974; Bloch et al., 1972, 1973; Orthoefer et al., 1976; Hyde et al., 1978; Vaughan et al., 1969
Guinea pig	0.08 (0.84±0.60 μm)	22-23 hours/day	12 months	12 months	Lowered growth rate in females. NOEL in other studies.	Alarie et al., 1973, 1975
Rat	2.37 (0.5±1.0 μm)	continuous	14 weeks	14 weeks	NOEL (lung function, blood chemistry, behavior).	Lawkowski et al., 1979
Rat	0.61 $SO_4^{=}$ (presumably most H_2SO_4) in auto exhausts	24 hours/day	7 days	7 days	Lower body weight.	Lee et al., 1976

*Source: Carson et al., 1981

NR = Not reported

24 hours/day, 7 days/week, for 14 weeks. There were no significant effects on behavior, as measured by spontaneous motor activity, lung function or blood acid-base chemistry. Lee et al. (1976) reported decreased body weight gain in female Sprague-Dawley rats (10/group) exposed to 0.6 mg $SO_4^=/m^3$ in auto exhausts 24 hours/day for 7 days. The $SO_4^=$ was, presumably, mostly in the form of H_2SO_4.

3.2. CHRONIC

3.2.1. Oral. Pertinent data regarding the chronic oral toxicity of sulfuric acid could not be located in the available literature.

3.2.2. Inhalation. Alarie et al. (1975) exposed groups of nine Cynomolgus monkeys of both sexes to atmospheres containing 0.09-0.99 mg H_2SO_4/m^3 in combination with varying amounts of fly ash and/or SO_2. At concentrations of 0.1-0.11 mg H_2SO_4/m^3, significant time-related increases in pulmonary resistance were reported for some combinations. No treatment-related effects were reported for any combination of exposures with a sulfuric acid concentration of 0.09 mg/m³.

Several investigators (Lewis et al., 1974; Bloch et al., 1972, 1973; Orthoefer et al., 1976; Vaughan et al., 1969; Hyde et al., 1978) exposed purebred female dogs (12/group, 20 controls) to atmospheres containing 0.09-0.11 mg H_2SO_4/m^3 and varying concentrations of SO_2 and auto exhaust 16 hours/day, 7 days/week, for up to 68 months, followed by observation for an additional 32-36 months. At the end of the 3-year observation period, there was significant ciliary loss, squamous metaplasia, air-space enlargement and nonciliated bronchiolar cell hyperplasia (Hyde et al., 1978).

Alarie et al. (1973, 1975) investigated the effects of chronic exposure of guinea pigs to sulfuric acid, with or without simultaneous exposure to 0.45 mg fly ash/m³. Groups of 50 male and 50 female Hartley guinea pigs

were exposed to atmospheres containing 0, 0.08, 0.10 or 0.30 mg H_2SO_4/m^3, 22-23 hours/day for 12 months. No effects were observed on survival, hematology or histology at any dose level. Exposure to sulfuric acid alone resulted in decreased growth rates in the females exposed to 0.08 or 0.10, but not 0.30 mg/m^3. No effects were reported in guinea pigs exposed to 0.08 mg H_2SO_4/m^3 and 0.45 mg fly ash/m^3.

Finklea et al. (1975a,b) estimated the threshold for increase in ambient air sulfate with increasing air pollution in terms of adverse effects in humans, based on epidemiological studies (Table 3-2). They estimated that exposure to as little as 0.01 mg H_2SO_4/m^3 for up to 10 years can result in an increased risk of chronic bronchitis in smokers. Total sulfate was determined in the atmosphere, but sulfuric acid is usually considered to be a major fraction of the measured sulfate. It is also often impossible to separate the effects of sulfuric acid from those of other pollutants (particularly O_3, NO_x, SO_2, HCl).

Occupational exposures have been associated with increased dental erosion, eye irritation and effects on the respiratory system (Tadzhibaeva and Gol'eva, 1976; El-Sadik et al., 1972; Williams, 1970; Malcolm and Paul, 1961; ten Bruggen Cate, 1958; Jones and Gamble, 1984; Gamble et al., 1984). The toxicity of sulfuric acid vapors to the human respiratory tract is well recognized. Williams (1970) observed a slight increase in bronchitis among workers exposed to sulfuric acid in a battery manufacturing plant compared with nonexposed workers at the same plant. Exposure levels had previously been estimated at 1.4 mg/m^3, which is sufficient to cause dental erosion in the exposed workers (Anfield and Warner, 1968). A higher incidence (36%) of bronchitis of apparently greater severity was noted by El-Sadik et al. (1972) in a group of 33 workers in two battery factories. The incidence of

bronchitis was 21% in unexposed controls (what size not reported). Some of the increased incidence of bronchitis in the exposed workers and the high incidence in controls appeared to be related to smoking habits. Dental infections and tooth discolorations were also observed in exposed workers and the incidence and severity appeared to correlate positively with duration of exposure.

Malcolm and Paul (1961) also reported severe tooth erosion among 160 acid-battery factory workers. The incidence and severity were more severe at concentrations of 3-16 mg/m^3 than at 0.8-2.5 mg/m^3. In some workers exposed to the higher levels the height of their incisors was decreased by as much as 50%. In a more recent investigation of the effects of sulfuric acid on the teeth, erosions were observed in workers exposed to atmospheres of 0-1.7 mg/m^3 (average 0.18 mg/m^3) (Jones and Gamble, 1984; Gamble et al., 1984). Exposure to 0.23 mg/m^3 for 4 months was sufficient to initiate erosion.

3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

3.3.1. Oral. Pertinent data regarding the teratogenicity or other reproductive effects of orally administered sulfuric acid could not be located in the available literature.

3.3.2. Inhalation. Pertinent data regarding the teratogenicity or other reproductive effects of inhaled sulfuric acid could not be located in the available literature.

3.4. TOXICANT INTERACTIONS

The toxicity of inhaled sulfuric acid is heavily influenced by, among other factors, the presence of other particulates in the air (Carson et al., 1981). Due to the hygroscopic nature of sulfuric acid, there is a tendency for sulfuric acid droplets to absorb water from the breath, creating larger

droplets that impact high in the upper respiratory tract where they cause less damage. When the sulfuric acid is absorbed to other particulates, it may be carried deeper into the respiratory tract.

Inhaled sulfuric acid mists are rapidly neutralized by NH_3 in the expired air (Barrow and Steinhagen, 1980). This neutralization is reduced when the sulfuric acid is absorbed to airborne particulates (Lawther, 1980).

Synergism has also been demonstrated between sulfuric acid and the pollutants normally found in auto exhausts (SO_2, ozone, metallic aerosols) (Carson et al., 1981). The specific interactions involved have not been fully elucidated and studies in humans have not consistently demonstrated the synergistic effects observed in experimental animals.

4. CARCINOGENICITY

4.1. HUMAN DATA

4.1.1. Oral. Pertinent data regarding the carcinogenicity of orally administered sulfuric acid could not be located in the available literature.

4.1.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled sulfuric acid could not be located in the available literature.

4.2. BIOASSAY

4.2.1. Oral. Pertinent data regarding the carcinogenicity of orally administered sulfuric acid could not be located in the available literature.

4.2.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled sulfuric acid could not be located in the available literature.

4.3. OTHER RELEVANT DATA

One study was located in which Escherichia coli (strain unspecified) were treated with 0.002-0.005% sulfuric acid solutions for 3 hours (Demerec et al., 1950, 1951). No increase in mutation frequency was observed.

4.4. WEIGHT OF EVIDENCE

IARC has not evaluated the risk to humans associated with oral or inhalation exposure to sulfuric acid. Data were not available regarding the carcinogenicity of sulfuric acid in humans or animals. Applying the criteria for evaluating the overall weight of evidence of carcinogenicity to humans proposed by the Carcinogen Assessment Group of the U.S. EPA (Federal Register, 1984), sulfuric acid is most appropriately designated a Group D - Not Classified chemical.

5. REGULATORY STANDARDS AND CRITERIA

The ACGIH (1980) has recommended a TLV of 1 mg/m³. This value is recommended to "prevent pulmonary irritation and injury to the teeth." The USSR has established an average maximum allowable concentration for the ambient air of populated places of 0.1 mg H_2SO_4/m³ with an allowable peak value of 0.3 mg/m³ (USSR State Committee of the Ministers for Construction, 1972). The ACGIH (1980) reports that both the USSR and Czechoslovakia have recommended a limit of 1 mg H_2SO_4/m³ for industrial exposures. The U.S. Occupational Safety and Health Administration has established an 8-hour TWA of 1 mg H_2SO_4/m³ (Code of Federal Regulations, 1981).

6. RISK ASSESSMENT

6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)

6.1.1. Oral. Pertinent data regarding the subchronic oral toxicity of sulfuric acid could not be located in the available literature.

6.1.2. Inhalation. The only reported effects of exposure to inhaled sulfuric acid were localized effects at the point of entry. Since an interim AIS is based on the absorbed dose, it is inappropriate to derive these numbers from local irritant effects. Based on studies with donkeys, which have respiratory systems anatomically similar to humans, subchronic exposure to atmospheric concentrations as low as 71 μg H_2SO_4/m^3 may produce a measurable decrease in the rate of bronchial mucociliary clearance (Schlesinger et al., 1978, 1979).

6.2. ACCEPTABLE INTAKE CHRONIC (AIC)

6.2.1. Oral. Pertinent data regarding the chronic oral toxicity of sulfuric acid could not be located in the available literature.

6.2.2. Inhalation. The only reported effects of chronic exposure to inhaled sulfuric acid are localized effects at the point of entry. Since an interim AIC is based on the absorbed dose, it is inappropriate to derive these values from the available data. There are some indications that tolerance to the irritant effects of sulfuric acid develops with repeated exposures (Hackney, 1978; Bushtueva, 1957). Thus, individuals may be able to tolerate chronic exposure to concentrations of sulfuric acid which would produce significant irritation during the initial exposure. Carson et al. (1981) estimated that "a lower exposure level that appears to be safe for man is in the range of 0.066-0.098 mg/m^3." They point out that this value may be influenced by a number of factors, including particle size, frequency and duration of exposure and synergistic effects.

This estimate correlates with the monkey data of Alarie et al. (1975) which showed a NOAEL of 0.09 mg/m³ even when sulfuric acid was combined with various combinations of fly ash and/or SO_2 which would be expected to exacerbate pulmonary irritation. In addition, the TLV has been set at 1 mg/m³; therefore, this suggested range incorporates a minimum of a 10-fold uncertainty factor for protection of more sensitive segments of the general population. Still troubling are the donkey data (Schlesinger et al., 1978, 1979), which show decreases in bronchial mucociliary clearance at 0.071 mg/m³. It is uncertain whether the donkey is unusually susceptible to sulfuric acid irritation or whether this study evaluated more sensitive endpoints than the other reports. The decrease in clearance could represent a metaplastic response of the bronchial epithelium to irritation, and this would not be detected as a deficit in pulmonary function. This deficit in clearance capacity, however, could compromise the ability to clear other respirable pollutants.

For these reasons, the range suggested by Carson et al. (1981) of 0.066-0.098 mg/m³ should be protective for exposures to sulfuric acid alone. As more data become available revision of this estimate may be required.

A CS was calculated for the respiratory tract effects observed in humans and animals and for the tooth erosion and etching that occurred in battery manufacturing workers exposed for as little as 4 months to sulfuric acid at 0.23 mg/m³. In the latter case, the exposure was expanded to continuous, assuming workers inhale 10 m³ of air during working hours on 5 days/week. An uncertainty factor of 10 was applied to convert from short-term to chronic exposure since it seems certain that prolonged exposure would result

in increased severity of the effect. A human MED of 0.16 mg/day resulted, corresponding to an RV_d of 6.7. Tooth erosion and etching were assigned an RV_e of 4. A CS of 26.8, the product of RV_d and RV_e, resulted.

6.3. CARCINOGENIC POTENCY (q_1^*)

6.3.1. Oral. Pertinent data regarding the carcinogenicity of orally administered sulfuric acid could not be located in the available literature.

6.3.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled sulfuric acid could not be located in the available literature.

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APPENDIX

Summary Table for Sulfuric Acid

	Species	Experimental Dose/Exposure	Effect	Acceptable Intake (AIS or AIC)	Reference
Inhalation					
AIS				ND[a]	
AIC				ND[a]	
Maximum composite score	human	0.23 mg/m³ occupational (0.16 mg/kg/day)[b] (RV_d = 6.7)	tooth etching and erosion (RV_e = 4)	26.8	Jones and Gamble, 1984; Gamble et al., 1984
Oral					
AIS				ND	
AIC				ND	

[a]See text for suggested maximum air concentration and rationale for not estimating acceptable intakes.

[b]An uncertainty factor of 10 was applied to convert from subchronic to chronic exposure; assumed: a worker breathes 10 m³ of air during working hours for 5 days/week.

ND = Not derived